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Biochemical, histological and histochemical studies of oat (*Avena sativa*) on hyperlipidemic rats.

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Abstract

Hyperlipidemia : is an elevation of lipids in the blood stream and these lipids include: fats, fatty acids, cholesterol, cholesterol esters, phospholipids, and triglycerides. Hyperlipidemia is associated with hepatic fat accumulation .

Material and methods:

Six groups (5rat/group) of female albino (*Rattus albinus)* were used. The 1st group used as control, in the 2nd group hyperlipidemia (25% fat & 2% cholesterol) was induced for 3 weeks only then sacrified , the 3rd group was hyperlipidemic rats for 3 weeks then left other 3 weeks without any additional treatment as a recovery period, the 4th group served as hyperlipidemic group for 3 weeks then treated with *Avena sativa* for another 3 weeks (200 g/Kg diet), the 5th group was hyperlipidemic (25% fat & 2% cholesterol) for 6 weeks and the 6th group served as hyperlipidemic rats for 6 weeks, and at the same time given *Avena sativa* in diet (200 g/Kg diet).

Results:

The biochemical parameters showed highly significant increase in body weight, serum glucose, AST, ALT, GGT, LDH, urea, creatinine, total protein, albumin, total lipids, cholesterol, triglycerides and LDL-cholesterol, while there was highly significant decrease in HDL-cholesterol.Many histopathological and histochemical changes were detected in liver tissue of the hyperlipidemic rats. Meanwhile, the treatment with oat ameliorated the biochemical parameters, histological and histochemical results

Conclusion:

It is recommend to use oat in diets for hyperlipidemic patients or those people who have hyperlipidemic family history.

Keywords:

Hyperlipidemia,Oat bran, Lipid profile, Albino rats, physiological parameters, histopathology and histochemistry.

Introduction

Hyperlipidemia (mainly increased level of total cholesterol (TC), triglycerides (TG) and low-density lipoprotein(LDL) cholesterol along with decrease in high-density lipoprotein(HDL) cholesterol) is the predictor of coronary artery disease (CAD). Hyperlipidemia is an important risk factor in the initiation and progression of atherosclerotic impasse **(Harrison *et al*., 2003).**

The hyperlipidemia-lowering effect of dietary plants has been well studied and various plants were shown to be helpful in lowering plasma lipid levels and encouraging safety profile. Dietary plants therefore are considered to be useful means to prevent disorders such as atherosclerosis **(Choudhary *et al*., 2005).**

One of the most important plants used as hyperlipidemia-lowering effect in the folk medicine in Egypt is oat herb (*Avena sativa*). Oat is considered as an important source of water-soluble fibers, have long been recognized as a potential cholesterol-lowering dietary component **(Davidson *et al*., 1991).**

The oat (*Avena Sativa*) is a species of cereal grains, and the seeds of this plant, are used

as food for people and animals, especially poultry and horses. Oat is the only cereal containing a globulin or legume-like protein, avenalins, as the major (80%) storage protein. Globulins are characterized by their water solubility, because of this property; oats may be turned into milk but not into bread. It is the more typical cereal proteins, such as gluten and prolamines. The minor protein of oat is a prolamine avenin. Oat protein is nearly equivalent in quality to soy protein which has been shown by the World Health Organization to be the equal to meat, milk, and egg protein **(Anderson *et al.*, 1990).**

Oat bran contains soluble fibers, such as β-glucan, that increase bile acid excretion and thus decrease serum cholesterol **(Reihner *et al.*, 1990).** The beneficial effects of oat products on the lipoprotein profile are ascribed to their soluble fiber compound, β-glucan **(Braaten *et al.*, 1994).**

β-glucan from oats is a non starch polysaccharide that is composed of β (1→4)-linked glucose units which are separated every 2-3 units by a single β-(1→3)-linked glucose unit **(Bell *et al.*, 1999).** β-glucan from barley **(Bourdon *et al.*, 1999)** or yeast **(Nicolosi *et al.*, 1999)** has also been shown to be hypocholesterolemic.

Oat is a source of many compounds that exhibit antioxidant activity. Vitamin E, phytic acid, significant quantities of phenolic compounds, and avenanthramides are the most abundant antioxidant in oat, flavonoids and sterols are also present **(Mensink and Katan, 1992; Peterson, 2001).**

So,this study aimed to evaluate the possible treatment and protective effect of *Avena sativa* in hyperlipidemic rats.

Material and methods

1-Experimental animals:

The present work was carried out on thirty mature female albino rats (150±20g). They were obtained from the Nile Company for Pharmaceutical and Chemical Industries. The experimental animals were randomly divided into six groups (5/group) and fed on rodent diet. The rats stayed for 3 weeks to adapt the place then the experimental steps were started.

2-Experimental design:

Six groups were used in this study each containing 5 female albino rats. The experiment lasts for 6 weeks.

1-The 1st group: served as control (C).

2-The 2nd group: hyperlipidemic rats (25% fat & 2%cholesterol)3 weeks only then they were scarified (H3).

3-The 3rd group: served as hyperlipidemic rats for 3 weeks then left other 3 weeks without any additional treatment as a recovery period (R).

4-The 4th group: served as hyperlipidemic rats for 3 weeks then treated with *Avena sativa* for 3 weeks (200 gm/Kg diet) (H3*A*).

5-The 5th group: included hyperlipidemic rats (25% fat & 2%cholesterol) for 6 weeks (H6).

6-The 6th group: served as hyperlipidemic rats for 6 weeks, and at the same time they were given *Avena sativa* in the diet(200gm/Kg diet) (H6*A*).

Each rat was weighted at the beginning and the end of the experiment and percentage of body weight changes were calculated.

Collection of rat's serum

At the end of the experiment, animals were decapitated and blood samples were collected from the retro-orbital plexus. The samples were collected in clean dry graduated centrifuge tubes and left for 20 minutes to clot, then centrifuged at 5000 rpm, for 15 minutes. Serum was separated and kept at -20ºC until analysis.

Serum glucose was estimated according to **Trinder (1984)**. Aspartate transaminase (AST) was performed according to **Bergmeyer (1978)**. Alanine transaminase (ALT) was determined according to **Breuer (1996)**. γ-Glutamyltransferase (γ-GT) was done according to **Szasz and Persijn (1974)**. Serum LDH (Lactate dehydrogenase) concentration was done according to the kinetic ultraviolet method of **Young (1990)**. Measurement of Serum Urea was done according to the method of **Patton and Crouch (1977).** Serum

creatinine was evaluated according to the method of **Jaffe (1980).** Serum total protein was performed by the method of **Tietz (1994)**.Serum albumin was done by the method of **Doumas *et al.* (1971).**

Total lipids was done by the method of **Kaplan (1984).** Serum total cholesterol(T.C) was performed according to **Henry *et al*. (1974) .** Serum triglycerides(T.G) were determined according to the method of **Fossati and Prencie (1982).** Serum high density lipoproteins cholesterol (HDL-Cholesterol) was done according to **Burstein (1970).** The concentration of low density lipoproteins cholesterol (LDL-Cholesterol) in serum was estimated by the equation used by **Friedewald *et al.* (1972)** as follow:

$$\text{LDL} - \text{cholesterol (mg/dl)} = \text{Total cholesterol} - \text{HDL cholesterol} - \left(\frac{\text{T.G}}{5}\right)$$

The histological and histochemical results:

Fresh specimens of liver were taken from the control and treated groups. The specimens were fixed in 10% neutral buffer formol and Carnoy's fluid for the histological and histochemical studies. Sections were then cut at 5µ thickness and stained by haematoxylin and eosin stain according to the method of **Drury & Wallington (1980),** periodic acid Schiff technique for demonstrating glycogen **(Pearse,1977).** Mercuric bromophenol blue method for detecting total protein **(Mazia *et al*., 1953).** Mallory's trichrome stain for demonstrating collagen fibers (**Pearse, 1977).**

Statistical analysis:

The data are expressed as means ± standard errors (SE). The (T) test was used to elucidate the differences between treated and control groups **(Snedecor and Cochran, 1980).** A difference was considered significant at $p< 0.05$ or $p< 0.01$.

Results

The percentage of body weight gain was highly significant increased ($P < 0.01$) in all treated groups. Concerning serum glucose level, the present data showed sever hyperglycemia ($P < 0.01$) in all the treated groups. (Table 1).

Results of the present study showed a high significant increase ($P < 0.01$) in AST, ALT, GGT and LDH activities in all the treated groups when compared with the control rats (Table 2).Also, high significant increase ($P < 0.01$) in serum urea and creatinine concentrations in all treated groups was detected(Table 3) with

high significant increase ($P < 0.01$) in serum total protein and albumin concentrations in all the treated groups when compared with the control one during the experimental period (Table 4). Globulin concentration showed insignificant change in all treated groups (Table 4). All treated groups also showed insignificant change in albumin/globulin ratio (A/G ratio) except in the group that fed hyperlipidemic diet for 6 weeks where it showed highly significant increase ($P < 0.01$) as compared with the control group (Table 4).

A high significant increase ($P < 0.01$) in serum total lipids was realized except in the group that treated with hperlipidemic diet for 3 weeks followed by oat for another 3 weeks which showed a significant increase ($P < 0.05$). Concerning liver total lipids highly significant increase ($P < 0.01$) was recorded (except in the group that treated with hperlipidemic diet for 3 weeks followed by oat for another 3 weeks) which showed highly significant decrease ($P < 0.01$). Cholesterol and triglycerides, showed highly significant increase ($P < 0.01$) in all the treated groups. HDL-cholesterol showed highly significant decrease ($P < 0.01$) in all the treated groups. Concerning LDL-cholesterol, highly significant increase ($P < 0.01$) was observed in all the treated groups. HDL/LDL ratio showed highly significant decrease ($P < 0.01$) in all the treated groups when compared with the control group (Table 5).

Hyperlipidemia for 6 weeks elevated all the studied parameters, while feeding oat for 3 weeks after stopping of fat diets recorded the lowest results in these parameters .

Normal histological pattern of liver tissue of a control rat was detected in figs. (1&2)Hyperlipidemic rats of groups H3 or H6 showed many pathological changes in

the liver tissue and the changes were more pronounced in rats of group H6. These changes include: highly distorted and ruptured endothelial lining of the blood vessels, increased lymphocytic infiltration in the portal area, haemolysed RBCs inside the blood vessels, degenerated and vacuolated hepatocytes with increased Kupffer and fatty cells (Figs. 3-5).A slight amelioration was noticed in liver tissue of rats of the recovery group (Figs. 6&7).

Noticeable signs of recovery were noticed in liver tissue of rats of groups H3A or H6A, but increased lymphocytic infiltration , haemolysed RBCs in the portal areas and distorted bile canaliculi with vacuolated hepatocytes were still observed (Figs.8,9,10 and 11).

Normal distribution of collagen fibers was observed in figs.(12&13).Increased collagen fibers were observed in liver tissue of groups H3 or H6, the recovery group and those treated with fats and Avena sativa for 3 or 6 weeks (Figs. 14,15,16,17).

Normal distribution of total protein in the hepatic tissue of a control rat was observed in figs (18&19).

Highly reduced total protein was observed in liver tissue of group H3 or H6 (Figs. 20 &21), but the R group showed a mild decrease. Meanwhile, normal total protein was observed in hepatocytes of liver tissue of groups H3A or H6A.

Normal distribution of polysaccharides was observed in figs. (22&23). Highly reduced polysaccharides were detected in liver tissue of groups H3 and H6 (Figs. 24 & 25), but thickened arterial walls showed increased stain affinity. Moderate stain affinity was detected in liver tissue of group R with normal stain affinity was observed in walls of the blood vessels (Figs. 26 & 27). Nearly normal glycogen content was noticed in liver tissue of groups H3A and H6A (Figs.28 & 29).

Concerning all the previous biochemical parameters, histological and histopathological changes it was found that using oat was better than only stop fats after hyperlipidemic diets without any additional treatment (recovery groups).

Table (1): Percentage of body weight change and Serum glucose level (mg/dl) in female albino rats after induction of hyperlipidemia and treating with oat (*Avena sativa*).

Group / Parameter		Control	3 weeks			6 weeks	
			Hyper L 3 W	Hyper L 3 W & Recov 3W	Hyper L 3 W then Oat 3 W	Hyper L 6 W	Hyper L & Oat 6 W
Body weight change (%)	Mean	5.50	12.92	10.84	8.18	13.58	12.30
	± SE	0.65	1.35	0.98	0.46	0.70	0.77
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	134.9	97.0	48.7	146.9	123.6
Glucos (mg/dl)	Mean	64.8	99.0	76.2	71.82	122.6	86.2
	± SE	1.7	2.4	1.7	0.926	1.03	1.1
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		--	52.7	17.59	10.83	89.19	33.0

Hyper L = Hyperlipidemia Recov = recovery 3W = 3 weeks 6W = 6 Weeks
N.S = non significan

Table (2): Aspartate transaminase (AST), Alanine transaminase (ALT) Glutamyl transferase (GGT) and Lactate dehydrogenase (LDH) activities in female albino rats after induction of hyperlipidemia and treating with oat (*Avena sativa*).

Group / Parameter		Control	3 weeks			6 weeks	
			Hyper L 3 W	Hyper L 3 W & Recov 3W	Hyper L 3 W then Oat 3 W	Hyper L 6 W	Hyper L & Oat 6 W
AST (U/L)	Mean	32.5	89.8	87.6	50.2	114.6	80.8
	± SE	0.8	0.96	2.99	0.96	2.3	0.82
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	176.3	169.5	54.4	252.6	148.6
ALT (U/L)	Mean	23.2	73.4	53.0	31.4	107.8	35.2
	± SE	1.4	1.8	1.1	1.2	2.3	2.1
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	216.3	128.4	35.3	364.6	51.7
GGT (U/L)	Mean	26.2	40.8	39.6	32.7	45.4	39.4
	± SE	1.85	0.6	0.57	0.675	1.35	0.570
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	55.7	51.1	24.8	73.2	50.3
LDH (U/L)	Mean	177.8	363.0	244.0	190.8	391.6	237.6
	± SE	2.8	1.9	2.26	0.96	4.3	9.0
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	104.1	37.2	7.3	120.2	33.6

Hyper L = Hyperlipidemia Recov = recovery 3W = 3 weeks 6W = 6 Weeks
N.s = non significant

Table (3): Serum urea and creatinine levels in female albino rats afte induction of hyperlipidemia and treating with oat (*Avena sativa*).

Group / Parameter		Control	3 weeks			6 weeks	
			Hyper L 3 W	Hyper L 3 W & Recov 3W	Hyper L 3 W then Oat 3 W	Hyper L 6 W	Hyper L & Oat 6 W
Urea (mg/dl)	Mean	30.4	43.04	42.6	39.6	46.5	43.0
	± SE	1.03	0.6	1.2	0.57	0.79	1.06
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	41.5	40.1	30.2	52.9	41.4
Creatinine (mg/dl)	Mean	0.688	1.039	0.971	0.932	1.206	1.150
	± SE	0.025	0.012	0.008	0.012	0.008	0.003
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	51.0	41.0	35.0	75.3	67.0

Hyper L = Hyperlipidemia Recov = recovery 3W = 3 weeks 6W = 6 Weeks
N.s = non significant

Table (4): Total protein, Albumin, Globulin concentrations an Albumin/Globulin ratio (A/G ratio) in female albino rats after induction of hyperlipidemia and treating with oat (*Avena sativa*).

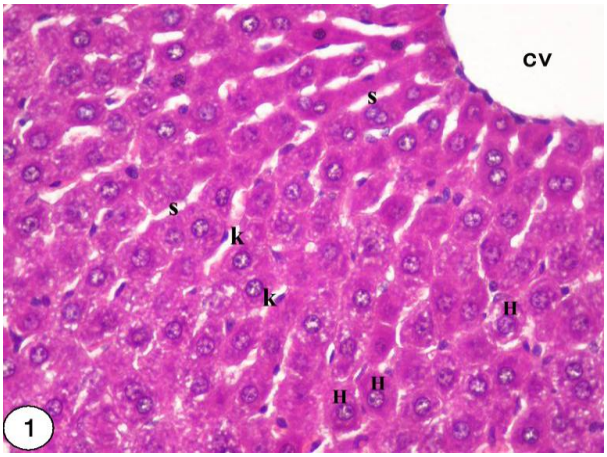
Group / Parameter		Control	3 weeks			6 weeks	
			Hyper L 3 W	Hyper L 3 W & Recov 3W	Hyper L 3 W then Oat 3 W	Hyper L 6 W	Hyper L & Oat 6 W
Total protein (g/dl)	Mean	6.7	8.46	8.0	7.74	9.12	8.1
	± SE	0.127	0.17	0.145	0.120	0.065	0.079
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	26.2	19.4	15.5	36.1	20.8
Albumin (g/dl)	Mean	4.32	5.7	5.4	4.9	6.5	5.52
	± SE	0.143	0.065	0.103	0.127	0.096	0.143
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	31.9	25.0	13.4	50.4	27.7
Globulin (g/dl)	Mean	2.38	2.74	2.56	2.84	2.6	2.58
	± SE	0.163	0.160	0.216	0.210	0.061	0.185
	P	-	N.S	N.S	N.S	N.S	N.S
% of change		-	15.1	7.5	19.3	9.2	8.40
A/G ratio	Mean	1.85	2.106	2.182	1.764	2.512	2.184
	± SE	0.177	0.128	0.249	0.187	0.094	0.214
	P	-	N.S	N.S	N.S	<0.01	N.S
% of change		-	13.8	17.9	- 4.6	35.7	18.0

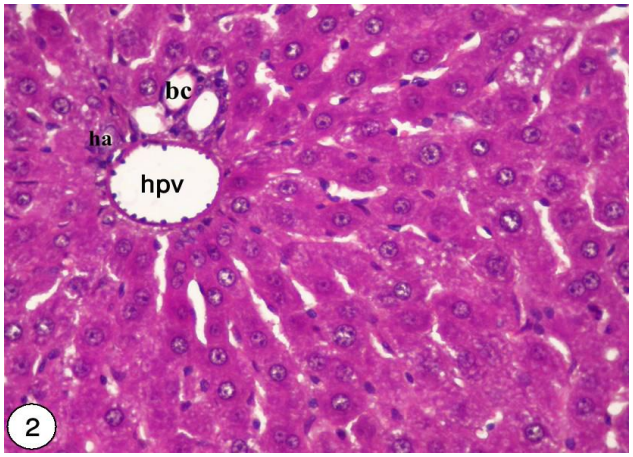
Hyper L = Hyperlipidemia Recov = recovery 3W = 3 weeks 6W = 6 Weeks
N.s = non significant

Table (5): The level of serum total lipids, liver total lipids, cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol and HDL/LDL ratio in female albino rats after induction of hyperlipidemia and treating with oat (*Avena sativa*).

Group / Parameter		Control	3 weeks			6 weeks	
			Hyper L 3 W	Hyper L 3 W & Recov 3W	Hyper L 3 W then Oat 3 W	Hyper L 6 W	Hyper L & Oat 6 W
Serum total lipids (g/l)	Mean	5.16	6.26	5.92	5.58	6.38	6.06
	± SE	0.125	0.075	0.041	0.11	0.17	0.05
	P	-	<0.01	<0.01	<0.05	<0.01	<0.01
% of change		-	21.3	14.7	8.1	23.6	17.4
Liver total lipids (g/l)	Mean	4.52	5.64	5.34	3.72	7.46	5.92
	± SE	0.21	0.13	0.125	0.14	0.16	0.151
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	24.7	18.1	- 17.6	65.04	30.97
Cholest-erol (mg/dl)	Mean	83.54	135.0	120.4	95.4	150.0	140.4
	± SE	2.31	0.79	0.570	0.570	0.790	1.03
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	61.5	44.1	14.1	79.5	68.06
Triglyc-erides (mg/dl)	Mean	93.2	140.4	124.6	115.4	161.2	139.2
	± SE	2.77	0.570	0.570	2.07	0.65	0.96
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	50.6	33.6	23.8	72.9	49.3
HDL-cholester-ol (mg/dl)	Mean	35.4	29.6	30.2	32.0	22.0	30.4
	± SE	0.57	0.75	0.74	0.79	0.79	0.83
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	- 16.3	- 14.6	- 9.6	- 37.8	- 14.1
LDL-Cholest-erol (mg/dl)	Mean	29.5	77.3	65.28	40.3	95.76	82.16
	± SE	2.39	1.39	0.167	1.32	1.50	1.54
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	162.0	121.2	36.6	224.6	178.5
HDL/L DL ratio	Mean	1.218	0.376	0.458	0.794	0.224	0.364
	± SE	0.089	0.0168	0.0129	0.0453	0.010	0.0175
	P	-	<0.01	<0.01	<0.01	<0.01	<0.01
% of change		-	- 69.1	- 62.3	- 34.8	- 81.6	- 70.1

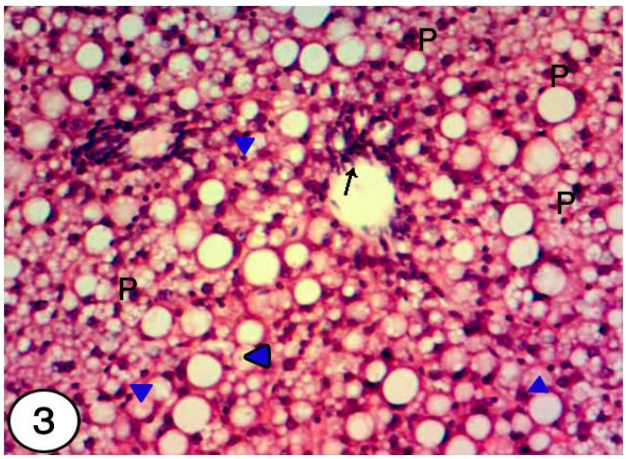
Hyper L = Hyperlipidemia Recov = recovery 3W = 3 weeks 6W = 6 Weeks
N.s = non significant

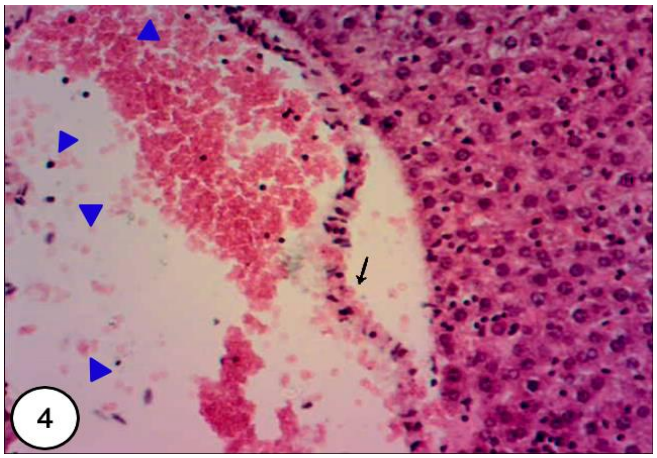




Figs.(1 & 2) showing photomicrographs of liver tissue of a control rat.
1- Showing the central vein (cv) , sinusoidal spaces (s) , kupffer cells (k) and hepatocytes (H).
(HX & E X100)

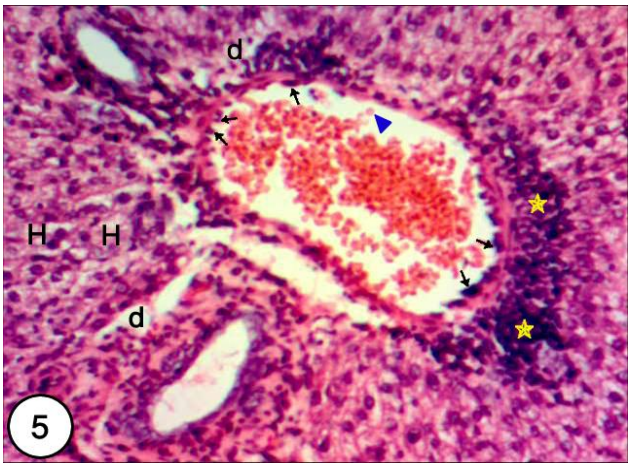
2- Showing the portal area which contains a branch of the hepatic portal vein (hpv) , bile canaliculi (bc) and a branch of the hepatic artery (ha).
(HX & E X100)



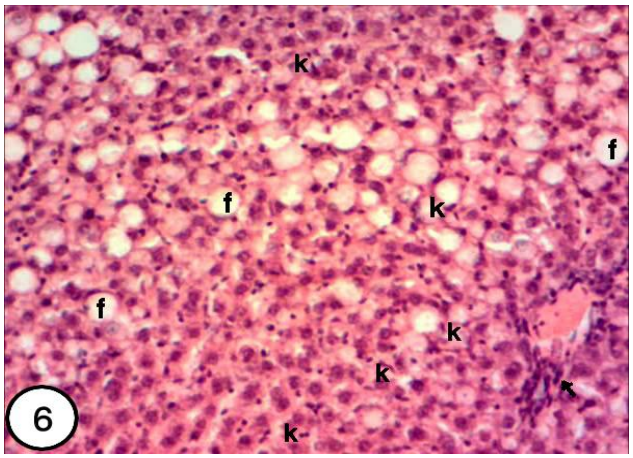


Figs . (3 ,4 & 5) showing photomicrographs of liver tissue of rats treated with fats (hyperlipidemia) for 3 weeks only.
3- Showing numerous fatty cells, lymphocytic infiltration around the distorted central vein (↗), lots of vacuolated hepatocytes (^) many pyknotic nuclei (p).

4- Showing delaminated endothelial lining of the central vein
(↗) and haemolysed RBCs (^).
(HX & E X100)



5 -Showing highly affected portal areas with enlarged nuclei of the endothelial lining of the branch of the hepatic portal vein (↗) , haemolysed RBCs (^) , highly distorted bile canaliculi (bc), increased lymphocytic infiltration ( ) , numerous vacuolated hepatocytes (H) and degenerated haepatocytes in the highly expanded empty spaces (d).



Figs . (6 & 7) showing photomicrographs of liver tissue of rats treated with fats for 3 weeks and left 3 weeks for recovery.

(HX & E X100)

6- Showing lymphocytic infiltration around the central vein (↗)، increased kuffer cells (k), fatty degeneration (F) with many vacuolated hepatocytes and haemolysed RBCs inside the central vein.

(HX & E X100)

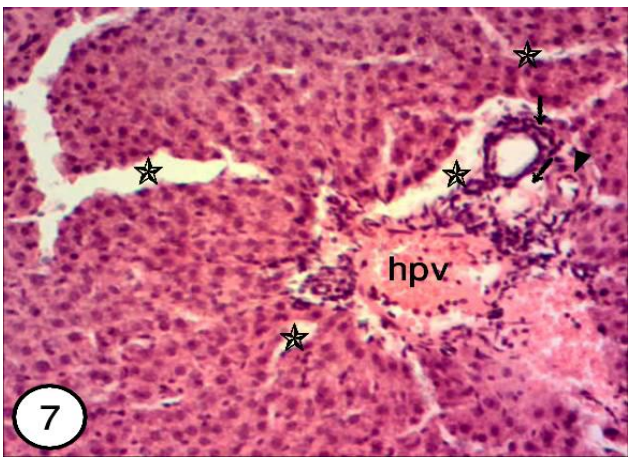


Fig. (7) Showing elongated and dilated hepatic portal vein (hpv), numerous empty spaces around the portal area and in between hepatocytes (☆) , distorted walls of bile canaliculi (↗) and branches of the hepatic arteries (^) with haemolysed RBCs inside hpv and increased lymphocytic infiltration in and around the portal areas. **(HX & E X100).**

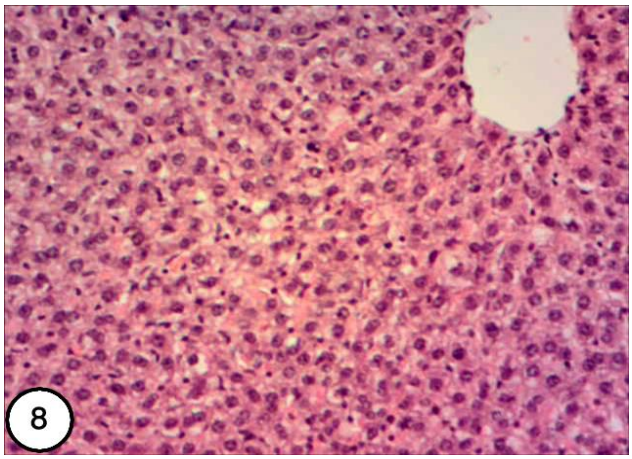


Fig.. (8) showing noticeable signs of recovery in the liver tissue of a rat treated with fats for 3weeks then 3weeks with *Avena sativa*.

Notice well developed hepatocytes and blood vessels, but lymphocytic infiltration is still observed in the portal area with haemolysed RBCs.

(HX & E X100).

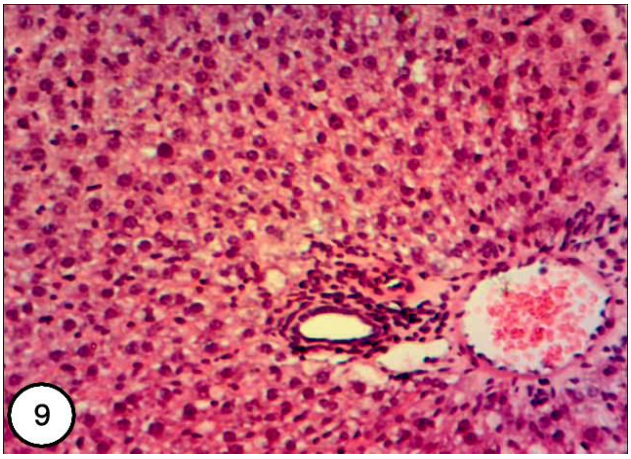


Fig.. (9) showing noticeable signs of recovery in the liver tissue of a rat treated with fats for 3weeks then 3weeks with *Avena sativa*.

Notice well developed hepatocytes and blood vessels, but lymphocytic infiltration is still observed in the portal area with haemolysed RBCs.

(HX & E X100).

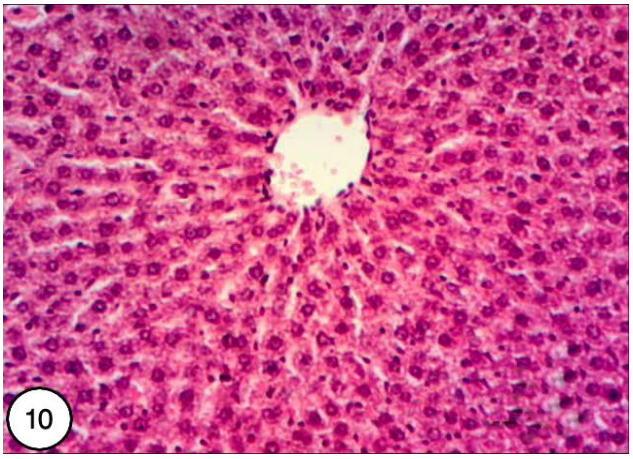


Fig. (10) showing photomicrographs of liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously. Notice: nearly complete recovery in the central area , while , the portal area showed ruptured endothelial lining of the dilated hpv and distorted and thickened walls of the bile canaliculi. Vacuolation was observed in some hepatocytes.

(HX & E X100).

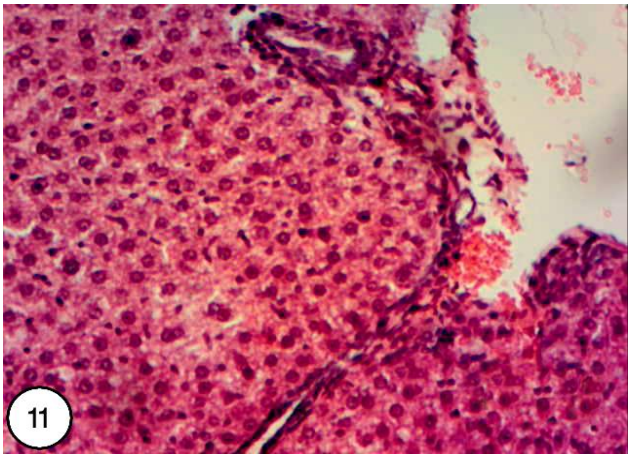


Fig. (11) showing photomicrographs of liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously. Notice: nearly complete recovery in the central area , while , the portal area showed ruptured endothelial lining of the dilated hpv and distorted and thickened walls of the bile canaliculi. Vacuolation was observed in some hepatocytes.

(HX & E X100).

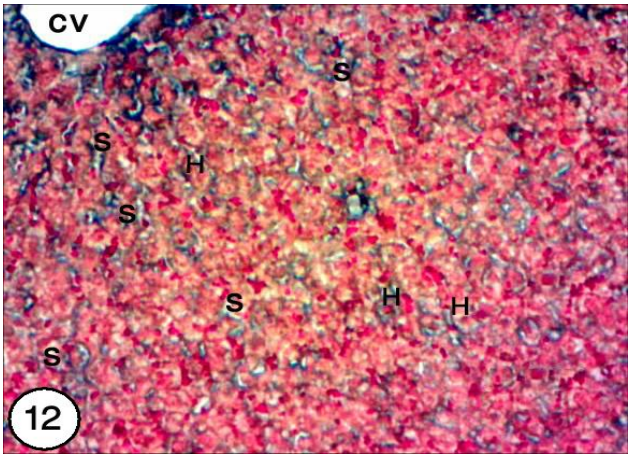


Fig.(12) showing normal distribution of collagen fibers in the liver tissue of a control rat. Notice thin collagen bundles supporting the central vein (cv) , hepatocytes (H) , sinusoidal spaces (S), hpv and walls of bile canaliculi.

(Mallory's trichrome stain X 100)

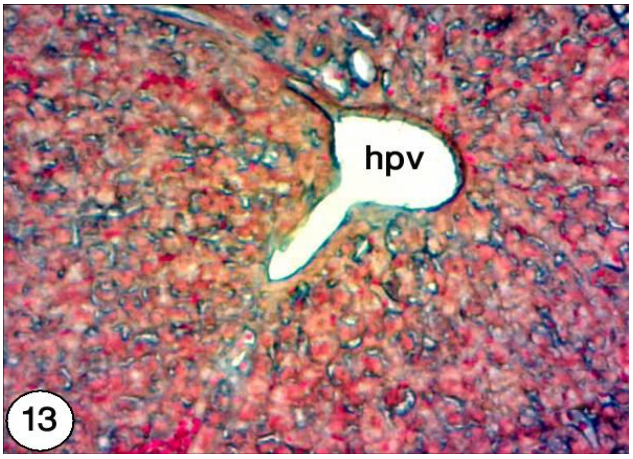


Fig.(13) showing normal distribution of collagen fibers in the liver tissue of a control rat. Notice thin collagen bundles supporting the central vein (cv) , hepatocytes (H) , sinusoidal spaces (S), hpv and walls of bile canaliculi.

(Mallory's trichrome stain X 100)

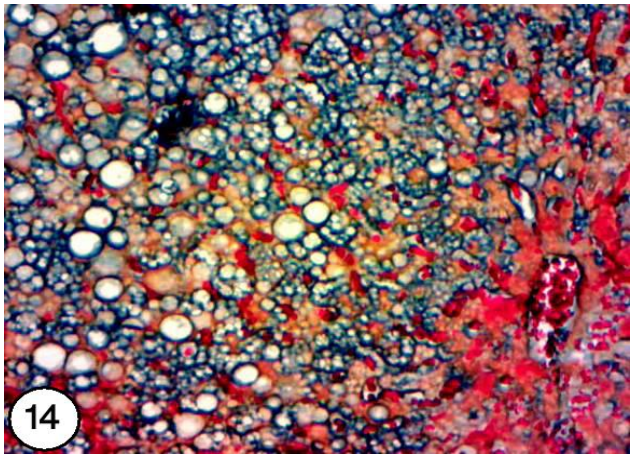


Fig.(14) showing increased collagen fibers in the liver tissue of a rat treated with fats (hyperlipidemia) for 3 weeks only. Collagen fibers increased around the hepatocytes , branches of the hepatic portal vein , hepatic artery , while , collagen fibers decreased in the wall of the central vein (↗) .

(Mallory's trichrome stain X 100)

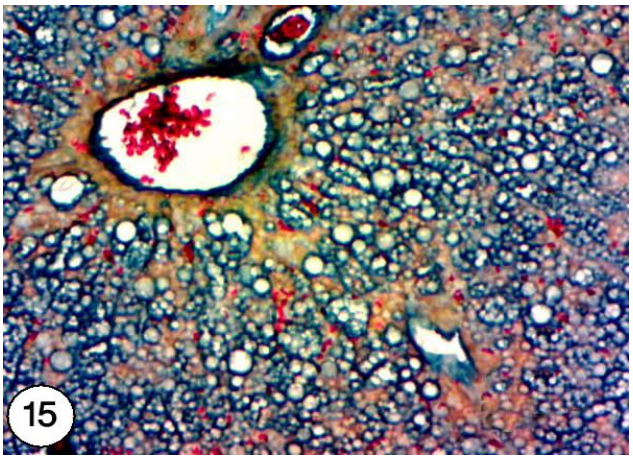


Fig.(15) showing increased collagen fibers in the liver tissue of a rat treated with fats (hyperlipidemia) for 3 weeks only. Collagen fibers increased around the hepatocytes , branches of the hepatic portal vein , hepatic artery , while , collagen fibers decreased in the wall of the central vein (↗) .

(Mallory's trichrome stain X 100)

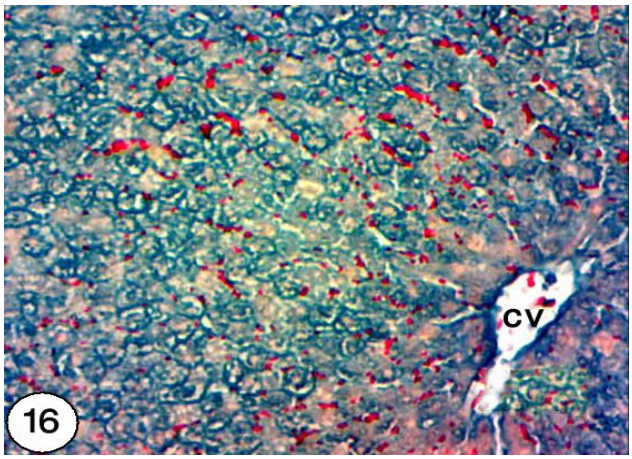


Fig.(16) showing distribution of collagen fibers in the liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously.

(Mallory's trichrome stain X 100)

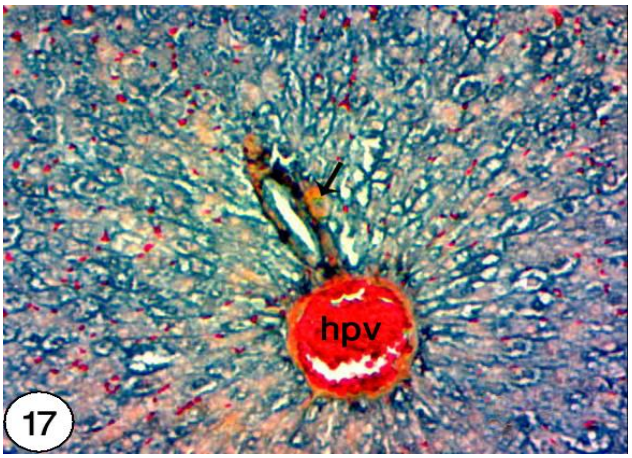


Fig.(17) showing distribution of collagen fibers in the liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously.

(Mallory's trichrome stain X 100)

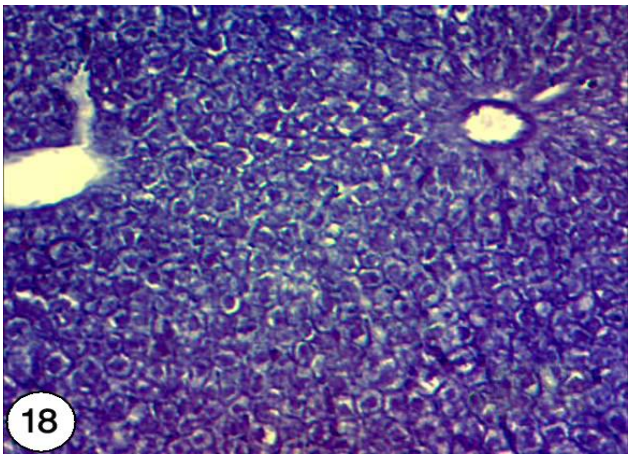


Fig.(18) showing normal distribution of total proteins in the central and portal areas in the liver tissue of a control rat.

(Mercuric bromophenol blue X 100)

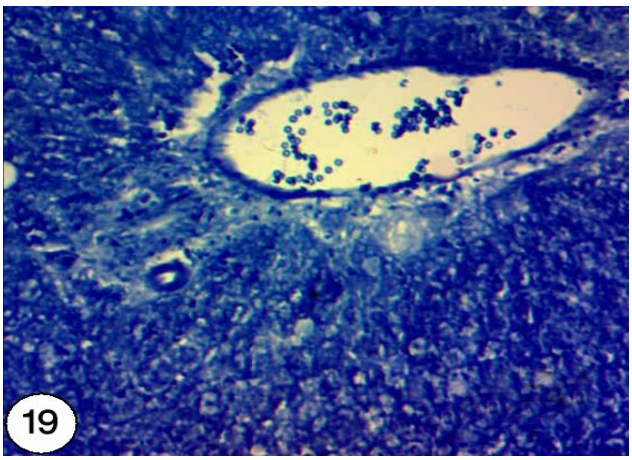


Fig.(19) showing normal distribution of total proteins in the central and portal areas in the liver tissue of a control rat.

(Mercuric bromophenol blue X 100)

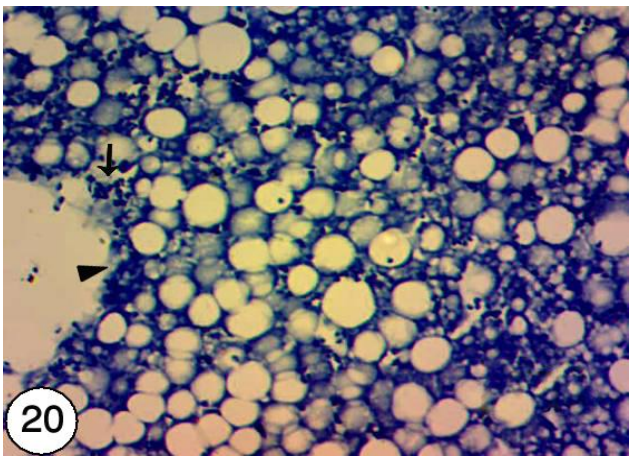


Fig.(20) showing highly reduced total proteins in hepatocytes, walls of the central vein ,hepatic portal vein , bile canaliculi in the liver tissue of rats treated with fats only for 6 weeks. Notice that congested and dilated sinusoidal spaces contained deeply stained RBCs (↗), the highly distorted central vein appeared faintly stained (^).

(Mercuric bromophenol blue X 100)

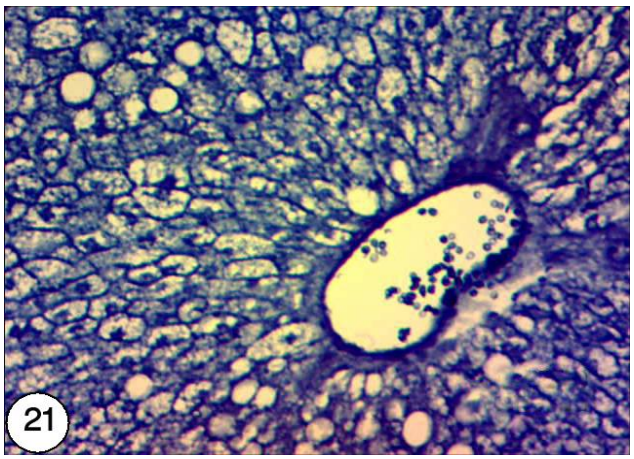


Fig.(21) showing highly reduced total proteins in hepatocytes, walls of the central vein ,hepatic portal vein , bile canaliculi in the liver tissue of rats treated with fats only for 6 weeks. Notice that congested and dilated sinusoidal spaces contained deeply stained RBCs (↗), the highly distorted central vein appeared faintly stained (^).

(Mercuric bromophenol blue X 100)

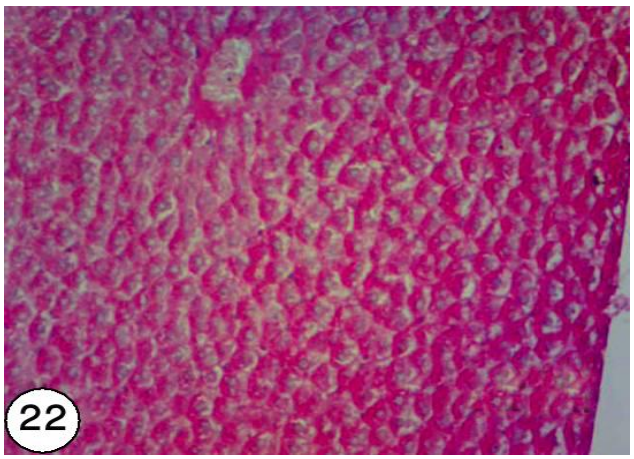


Fig.(22) showing normal glycogen content in the central and portal areas of liver tissue of a control rat.

(PAS X 100).

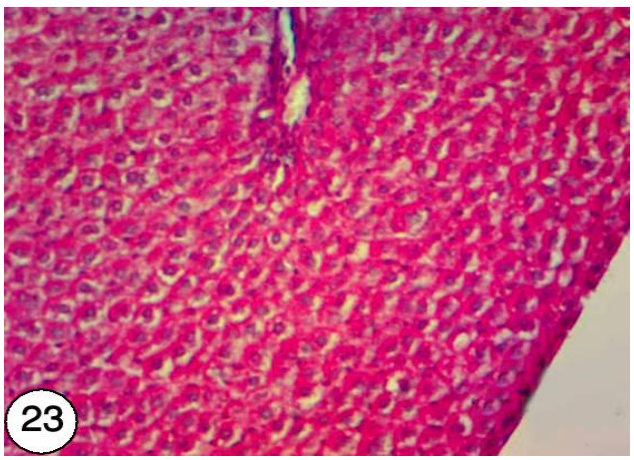


Fig.(23) showing normal glycogen content in the central and portal areas of liver tissue of a control rat.

(PAS X 100).

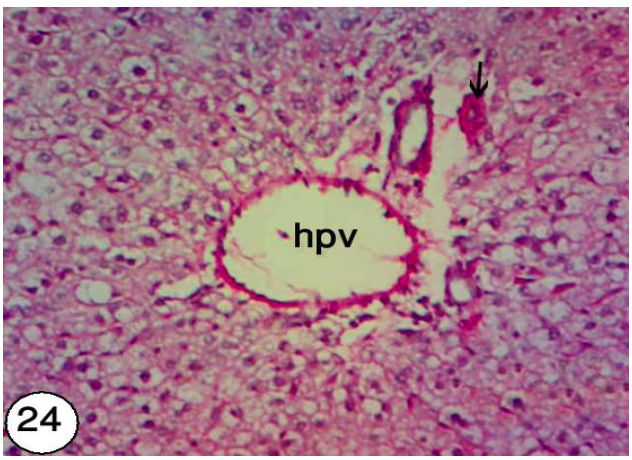


Fig.(24) showing glycogen distribution in the liver tissue of a rat treated with fats only for 6 weeks .

Notice : Highly depleted hepatocytes in the portal and central areas, endothelia lining of the central vein with increased stain affinity in the thickened arterial wall (↗). **(PAS X 100).**

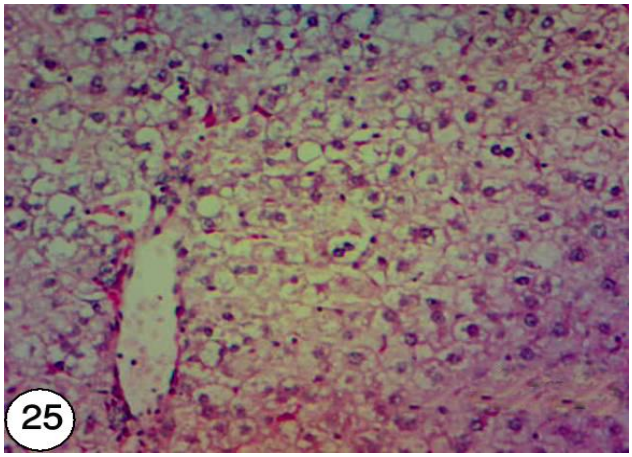


Fig.(25) showing glycogen distribution in the liver tissue of a rat treated with fats only for 6 weeks .
Notice : Highly depleted hepatocytes in the portal and central areas, endothelia lining of the central vein with increased stain affinity in the thickened arterial wall (↗). **(PAS X 100).**

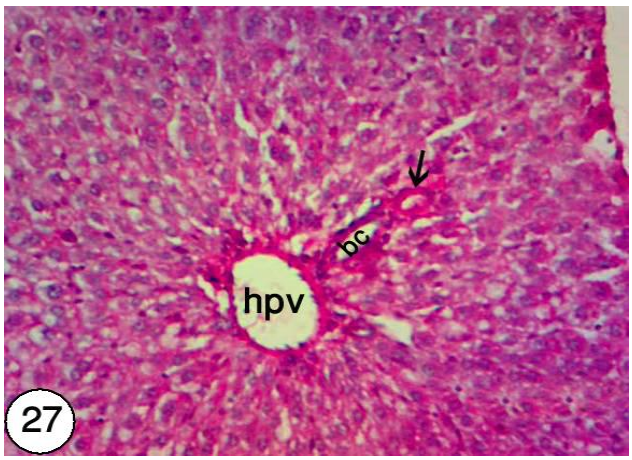


Fig.(27) showing glycogen distribution in the liver tissue of a rat treated with fats for 3 weeks and left 3 weeks for recovery.
Notice: Moderate stain affinity of glycogen in hepatocytes in the portal and central areas with normal stain affinity in the walls of hpv, artery (↗) and bile canaliculi (bc).

(PAS X 100).

Figs.(29) showing glycogen distribution in the liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously
Notice: Nearly normal glycogen content in the central and portal areas.

(PASX 100).

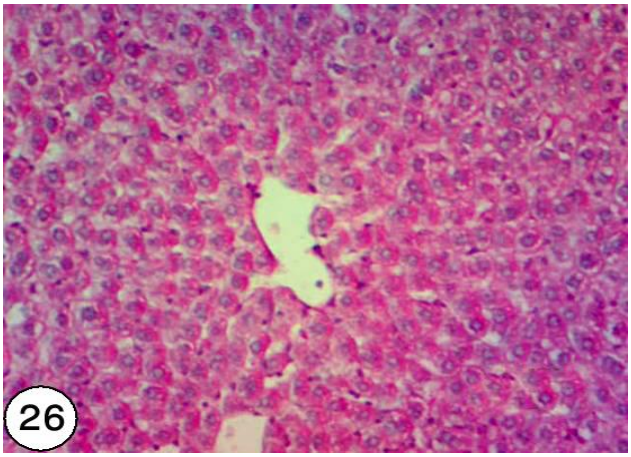
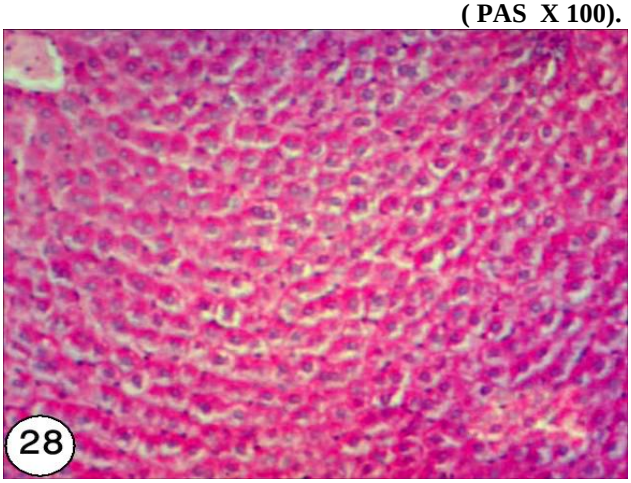
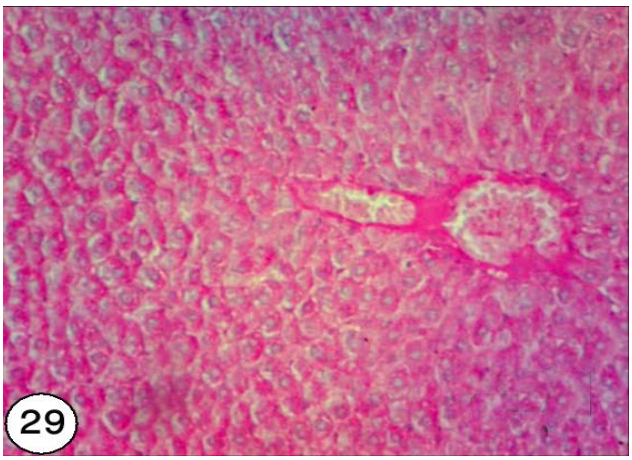


Fig.(26) showing glycogen distribution in the liver tissue of a rat treated with fats for 3 weeks and left 3 weeks for recovery.
Notice: Moderate stain affinity of glycogen in hepatocytes in the portal and central areas with normal stain affinity in the walls of hpv, artery (↗) and bile canaliculi (bc).

(PAS X 100).



Figs.(28) showing glycogen distribution in the liver tissue of a rat treated with fats and *Avena sativa* for 6 weeks simultaneously
Notice: Nearly normal glycogen content in the central and portal areas. **(PASX 100).**



Discussion:

In recent years, obesity has been considered as one of the main causes of cardiovascular disease, diabetes, and cancer .An increase in the prevalence of obesity and overweight has been widely reported**(Brown and Siahpush, 2007)**.

Body weight:

In the present study, although all groups recorded highly significant increase ($P < 0.01$) in the body weight(except the group which treated with oat and has free fat diet)where it showed less body weight gain (it recorded 48.7% while hyperlipidemic group recorded 134.9%), it also was better than recovery group which recorded 97.0% in comparison with control rats (Table 1).

This may be because its dietary fibers provide less energy as a substitute for nutrients in diets and prolong satiety **(Lairon, 2007).** In addition, they may slow down the absorption rate of nutrients and bind with dietary fat **(Pereira and Ludwig, 2001).**

Consequently, the foods rich in dietary fibers may assist body weight control. Also, it was reported that high-fiber and low-fat diet may prevent overweight or obesity and cardiovascular disease in normal adults **(Howarth *et al*., 2005).**

Dietary fibers can affect satiety and promote weight loss by several mechanisms such as lowering serum insulin,decreasing food intake since insulin stimulates hunger , slowing gastric emptying, contributing to feeling of fullness and they may increase rates of dietary thermogenesis compared to low-fiber food **(Anderson,1990).**

Glucose level:

In the present study using oat with the diet for 3 weeks causes reduction in serum glucose level where it recorded 10.83%, while hyperlipidemic group for 3 weeks recorded 52.7%. Glucose and insulin decreased as the β-glucan content increased; the highest β-glucan content resulted in a significant decrease in glucose **(Behall *et al*., 2006)**. Delayed or reduced carbohydrate absorption from the gut and not the effects of fermentation was suggested as the mechanism of action of oat β-glucan in postprandial glucose metabolism **(Wood *et al*., 2000).**

Liver and heart functions:

In the present results,rats fed oat (which contains beta-glucan) obviously decreased the levels of ALT and AST in the liver that injured due to feeding high fat diet for 3 weeks. It is concluded that oat β-glucan can relieve liver injury in hyperlipidemic rats **(Suping *et al*., 2009)**. **Howarth *et al*. (2005)** reported that high-fiber and low-fat diet may prevent cardiovascular disease in normal adults.

Kidney function:

In the present investigation, highly significant increase ($P < 0.01$) in serum urea and creatinine was observed in all treated groups. Dyslipidemia is a known risk factor for cardiovascular diseases and may associate with renal injury. In presence of severe hyperlipidemia progressive accumulation of lipids in the kidney drives the cascade of events that leads to impaired organ function **(Stevenson and Kaysen, 1999; Majumdar and Wheeler, 2000).** Lipid peroxidation, mediated by free oxygen radicals, is believed to be an important cause of destruction and damage to cell membranes, since polyunsaturated fatty acids of the cellular membrane are degraded by this process with consequent disruption of membrane integrity **(Garcia *et al*., 1997)**. Several mechanisms were proposed for the protective effect of β-glucan, one of these mechanisms is related to antioxidant capacity of this molecule **(Babincova *et al*., 2002 and Krizkova *et al*., 2003)**. Impairment in renal functions observed in the present study was improved by oat (β-glucan) treatment. One of the proposed mechanisms for β-glucan protection in renal function was free radical scavenging activity **(Sener *et al*., 2006)**. Membrane peroxidation can lead to changes in membrane fluidity and permeability, and also to enhanced rates of protein degradation, these will eventually lead to cell lysis **(Garcia *et al*., 1997)**. **Sener *et al*. (2006)** reported that

the antioxidant β-glucan inhibits elevation of serum urea and creatinine and reverses it back to the control levels. **Reiter *et al*.(2001)** proposed that impairment of antioxidant defense mechanisms could permit enhanced free radical induced renal tissue damage. They added that β-glucan, which is known to have immunomodulatory effects has antioxidant properties.

Protein profile:

Increased serum total protein and albumin observed in the present study may be due to increased amino acids synthesis. This increase in protein synthesis may be due to the increase in the amount and availability of mRNA **(Peavy *et al*., 1985)** , increase in translation factor**(Wool *et al*., 1986)** and increase in ribosomal protein synthesis as a result of hyperlipidemia. **Adam *et al*. (2007)** suggested that diets high in vegetable protein may permit safe weight loss in overweight or obese patients with chronic kidney disease (CKD). Oat protein is nearly equivalent in quality to soy protein which has been shown by the World Health Organization to be the equal to meat, milk, and egg protein **(Anderson *et al*., 1990).** In rats with obesity-related nephropathy, a soy diet, improved renal function, proteinuria, glomerulosclerosis, and interstitial fibrosis **(Trujillo *et al*., 2005)**.

Lipid profiles:

The present results showed that oat significantly lowered serum total lipids and recorded highly significant decrease in liver total lipids. This may be due to β-glucan have a lower molecular weight (BG370) rather than higher molecular weights (BG 1450 and BG 730) which significantly affected the levels of total lipid in serum **(Wilson *et al*., 2004)**.

Dietary fibers, as oat may affect postprandial lipid metabolism by several mechanisms such as: (1) altering gastric emptying ,(2) influencing intestinal transit time secretion , (3) modifying pancreatic secretion or pancreatic enzyme activity (4) acting on micelle formation **(Deshaies *et al*.,1990).**

The present results revealed that oat reduces the cholesterol level in rats that fed a high fat diet, where it recorded 14.1%, while hyperlipidemic group recorded 61.5%, and recovery group recorded 44.1%. These results may be due to oat's fibers operating on cholesterol through completely different mechanisms, such as alter the metabolism of bile acids and sterol faecal excretion, the reduction of nutrient absorption or cholesterol synthesis may be inhibited by short-chain fatty acids produced by fermentation in the colon **(Moundras *et al*., 1994 ; Olleros *et al*., 1999).**

Beneficial effects of high dietary fiber intake as oat β-glucan in patients with hyperlipidemia were detected with a significant decrease of triglycerides and LDL-Cholesterol **(Chandalia *et al*., 2000).**

The present results elucidated that HDL-Cholesterol concentration showed highly significant decrease ($P < 0.01$) in all the treated groups, but group that treated with oat and fed free fat diet showed the best results where it recorded -9.6% ,while hyperlipidemic group recorded -16.3% , also it recorded better result than recovery group which recorded -14.6% as compared with the control group.

β-glucan from oat decreased serum LDL cholesterol and increased bile acid excretion which promotes bile acid synthesis from cholesterol, so it increase LDL cholesterol uptake in the liver**(Theuwissen and Mensink,2008). Yu *et al*.(2002**) reported that radical scavenging of oat components could reacts with radicals by donating protons (free radical quenching), radical addition, redox reaction (electron transfer) and radical combination **.**

The oat which is a species of cereal grain has potent beneficial health effects in reducing LDL-cholesterol and should be included in the prudent diet of individuals with hyperlipidemia **(Al-Rawi, 2007).**

The histopathological study:

Rats treated with fats for 3 or 6 weeks showed many pathological changes in the liver tissue. These changes were more pronounced in liver tissue of group H6.

These changes include: ruptured endothelial lining of the blood vessels, enlarged nuclei of the endothelial lining, increased lymphocytic infiltration in the portal area, haemolysed RBCs inside the blood vessels with degenerated and vacuolated hepatocytes and fatty cells. The hepatic portal areas lost their normal architecture and contained highly distorted blood vessels and bile canaliculi.

Hyperlipidemia is known to enhance the risk of fatty liver disease **(Festi *et al.*, 2004)** and carcinogenesis which is associated with hydroxyl radical formation **(Tseng *et al.*, 1996).** Nuclear changes observed in the present study are in accordance with the results of **Nayana and Janardhanan (2000)** who stated that reactive oxygen species (ROS) such as superoxide anions (O_2), hydrogen peroxide (H_2O_2), hydroxyl radical (OH) and nitric oxide (NO) are directly or indirectly involved in DNA damage leading to mutations.Low grade inflammation, endothelial dysfunction and decreased fibrinolysis were associated with increased cardiovascular risk caused by hyperlipidemia **(Pidker *et al.*, 2002).**

In the present study, rats of group R showed a slight improvement in the hepatic tissue, but noticeable sings of recovery were detected in liver tissue treated with fats for 3 weeks and then treated with *Avena sativa* for 3 or 6 weeks. Well developed blood vessels and hepatocytes were detected with the exception of increased lymphocytic infiltration and haemolysed RBCs in the portal area. Hyperlipidemia leads to fatty liver disease which is associated with hydroxyl radical formation, these radicals may be responsible for degenerative changes observed in the liver of hyperlipidemic rats. Several authors showed that oats contained sevnumerous beneficial components such as vitamin E (α-tocopherol), phenolic acids, flavonoids, sterols and β-glucan **(Nie *et al.*, 2006; Theuwissen and Mensink, 2008).**

Several authors mentioned that oats contained β-glucan which is shown to posses potent antioxidant and free radical scavenging capabilities **(Bobeck and Calbavy, 2001; Kenneth and Hunter, 2004).**

Results of the present study showed increased stain affinity of collagen fibers in liver of rats of groups H3, H6 and R, especially around the haepatocytes, walls of hepatic portal veins and the arterial walls with decreased stain affinity in the wall of the central vein.

Horn *et al.* (1985) declared that the presence of collagen in the presinusoidal spaces might affect the blood supply to liver cells and would reduce the exchange of metabolitesmay cause hepatocellular dysfunction and necrosis.Liver of rats of group H3*A* & H6*A* showed increased collagen fibers in walls of blood vessels, hepatocytes and sinusoidal spaces.

Rousovan *et al.* (1992) declared that the increase in collagen fibers may be due to increased interstitial tissue and the white fibers under the effect of physical factors, but **Hassan *et al.* (1988)** reported that increased collagen fibers may lead to increase the defense reaction against toxic materials.

The histochemical study:

Highly reduced total protein was detected in liver tissue of groups H3 & H6, but nearly normal protein content was realized in hepatocytes of liver tissue of group R, but a mild decrease was noted in walls of the blood vessels and bile canaliculi.

Nearly normal protein content was observed in liver tissue of groups H3*A* and H6*A.* Some vacuolated hepatocytes and fatty cells were negatively stained with deeply stained RBCs inside the congested central vein and the hepatic portal vein.According to **El-Banhawy *et al.* (1986)** decreased protein content in the liver tissue may be due to increased action of lytic enzymes.

Highly decreased PAS positive materials were detected in liver tissue of groups H3 & H6, but thickened arterial walls were deeply stained.

Most hepatocytes of liver tissue of group R, showed normal content of PAS positive materials.

Signs of improvement in liver tissue of rats treated with fats for 3 or 6 weeks and *Avena*

sativa was accompanied by restored glycogen content in the liver tissue.

Highly affected glycogen content observed in this study in liver of rats treated with fats may be due to altered insulin levels and insulin sensitivity or insulin resistance due to obesity. The present results are in parallel with those recorded by **Han *et al.* (2007).**

Conclusion & recommendations:

Results of the present study showed that oat has hypolipidemic action specially when used with free fat diet for treating hyperlipidemia.So,we recommended to use it in treatement hyperlipidemic patients.

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دراسات بيوكيميائية وهستولوجية وكيميانسيجية لنبات الشوفان على الجرذان المصابة بزيادة الدهون

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إستهدفت هذه الدراسه إيضاح الدور الوقائى لنبات الشوفان ضد الأخطار الناتجه عن زيادة الدهون على بعض المعايير البيو كيميائية وقسمت هذه الحيوانات إلى المجموعات التاليه:

١-المجموعة الأولى:- استخدمت كمجموعه ضابطه.

٢-المجموعة الثانية:- مجموعة الجرذان ا لمصابة بالدهون(٢٥% دهون& ٢% كولستيرول) لمدة ٣ أسابيع فقط ثم تم ذبحها.

٣-المجموعة الثالثة:- مجموعة الجرذان المصابة بزيادة الدهون لمدة ٣ أسابيع ثم تركت ٣ أسابيع بدون أى علاج إضافى كفترة إستشفاء.

٤- المجموعة الرابعة:-الجرذان المصابة بزيادة الدهون ٣اسابيع ثم عولجت بنبات الشوفان (٢٠٠جم / كيلوجرام من وزن الجسم)لمدة ٣ اسابيع اخرى.

٥-المجموعة الخامسة: المجموعة المصابة بالدهون لمدة ٦ أسابيع (٢٥% دهون& ٢% كولستيرول).

٦-المجموعة السادسة:- مجموعة الجرذان المصابة بزيادة الدهون وفى نفس الوقت تناولت الشوفان لمدة ٦ أسابيع (٢٠٠جم / كيلوجرام من وزن الجسم).

ولقد أوضحت نتائج هذا البحث أن معاملة الجرذان بالدهون لمدة ٣ أو ٦ أسابيع أو حتى بعد التوقف عن تعاطى الدهون لمدة ٣ أسابيع له آثارسلبيه عديدة تتمثل فى زيادة معدل كلا من: وزن الجسم- نسبة السكر فى الدم-وظائف الكلى- وظائف الكبد وإنزيمات القلب- البروتين الكلى- الألبيومين ومجموعة الدهون والكوليسترول والدهون الثلاثيه بالدم ، كما تشتمل أيضا على النقص الشديد فى HDL-Cholesterol وصاحب كل ذلك وجود عدد كبير من التغيرات النسيجية والكيميانسيجية فى كبد الجرذان البيضاء أما المعاملة بنبات الشوفان فقد أدت الى ظهورتحسن ملحوظ فى المعايير البيوكيميائية والهستولوجية والهستوكيميائية ولهذا ينصح بإستخدام نبات الشوفان فى طعام مرضى زيادة الدهون أومن لهم تاريخ عائلى لمرض زيادة الدهون